homeostasis. While these tissues move protons, bicarbonate, and other ions transepithelially with a suite of pumps and transporters, how these proton-secreting cells (PSCs) arise developmentally is unknown. Here, we identify a cell type in the *X. laevis* larval that expresses a collection of transporters strikingly similar to those observed in the mammalian kidney, including "kidney"-specific isoforms of the H+V-ATPase and Cl−/HCO3− antiporters related to pendrin and AE1. We show that the transcription factor Foxi1 drives expression, perhaps directly, of some of these genes in a manner similar to the mammalian kidney and epididymis. Moreover, ectopic Foxi1 expression is sufficient to promote the formation of PSC precursors and their intercalation, thus mediating the early specification and morphogenesis of PSCs. We also show that PSCs form in the skin as different subtypes that strongly resemble, in both gene expression and protein localization, the alpha- and beta-intercalated cells of the cortical collecting duct, cells specialized for proton or bicarbonate secretion in the mammalian kidney. Finally, we show that the grainyhead-related transcription factor ubp1L promotes the differentiation of beta-like PSCs and represses alpha-like PSCs. These results hint at a mechanism for PSC specification in vertebrates and shed light on how transport epithelia acquire cellular and functional heterogeneity.

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Program/Abstract # 235
A novel pRb protein network controlling *C. elegans* organogenesis
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Studies on the *C. elegans* Retinoblastoma protein (pRb) ortholog, LIN-35, have uncovered a wide range of cellular and developmental functions that are mechanistically distinct from the canonical role of pRb family proteins in cell cycle control. Correspondingly, mammalian pRb, along with its E2F binding partners, are thought to inhibit tumor progression at a number of distinct steps in addition to their role in repressing cell proliferation. Using a combination of genetic, bioinformatical, and molecular approaches, we have uncovered a novel LIN-35-associated network that regulates development of the *C. elegans* pharynx. More specifically, this pathway controls an early step of pharyngeal morphogenesis that involves stereotypical changes in epithelial cell shape and polarity. Proteins within this network include several conserved ubiquitin-ligase components, a number of transcriptional regulators, and may also involve interactions with the microtubule cytoskeleton. These studies may provide insight into a suggested role for pRb incontrolling cancer metastasis and more generally can shed light on the mechanistic basis for tumor suppression by pRb family members.

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Program/Abstract # 236
Large scale analysis of gene expression in the murine embryonic lung
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In order to obtain a comprehensive framework for understanding and investigating the genetic program that controls mammalian lung development, we have systematically mapped gene expression in the embryonic mouse lung by in situ hybridization. Our analysis of the expression patterns of ~2500 genes has given us insights into the patterning of the lung, the control of the tissue and cell differentiation within the embryonic lung, and numerous molecular markers for those processes. These studies will provide the first global view of the temporal and spatial gene expression program for the formation of the mammalian lung. Many of the genes have specific expression patterns that suggest important and specific roles in embryonic lung morphogenesis. We will demonstrate how these studies focus these subsequent analyses and provide molecular markers to better understand changes to the lung observed in these mutants. We believe these methods can be generalized to study the changes caused by genetic defects, disease, toxins and drugs.

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Program/Abstract # 237
Regulation of airway shape by SPROUTY-mediated control of oriented cell division
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The sizes and shapes of the epithelial tubes that comprise organs such as lung, kidney, and vasculature are critical for their function. Here we investigate the mechanisms that control airway morphogenesis and show that early in lung development, airway shape is a function of the orientation of planar cell division. In normal airways, a large proportion of epithelial cell divisions are oriented parallel to the airway longitudinal axis, whereas this distribution is randomized when RAS-regulated ERK1/2 signaling is increased, leading to shorter and wider airways. We have developed a mathematical model that predicts epithelial tube shape from the distribution of mitotic spindle angles during development, and show that the abnormal shapes of airways in which ERK1/2 signaling is increased can be accounted solely for by the observed alterations in mitotic spindle orientation. Our data reveal that regulating ERK1/2 signaling is essential to ensure appropriate oriented planar cell division, and demonstrate the importance of the negative regulators of this signaling pathway that are encoded by the Sprouty genes for maintaining the normal airway morphogenesis program.

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Program/Abstract # 238
Sprouty gene function in otic placode induction
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Sprouty (Spry) genes encode antagonists of receptor tyrosine kinase signaling, including Fibroblast Growth Factor (FGF) signaling. We have found that in mouse embryos missing both the Spry1 and Spry2 genes ($Spry1^{-/-}$; $Spry2^{-/-}$ double mutants) the otic placode is expanded. Consistent with a role in otic placode induction, we have found that both Spry1 and Spry2 are co-expressed in the pre-otic ectoderm and underlying mesenchyme. FGFs have been shown to induce otic placode formation in multiple species including mouse, chick, and zebrafish. In the mouse, double mutant combinations of Fgf3 and Fgf10 or Fgf8 and Fgf3 result in the absence or dramatic